Egyptian Nutrition Society-Special Issue :
The First International Confrence of Nutrition,Hurghada,April 2017

Effect of Evening Primrose Seed Oil Against Stannous Chloride Induced Toxicity in Male Rats

Hanaa. F. El Mehairy and Mostafa, M.Y.A.

Home Economics Department, Faculty of Specific Education, Mansoura University, Egypt.

Abstract

Evening primrose seed oil *(Oenothera biennis)* has many activities including anti-oxidant, and anti-inflammatory. The present study was carried out to investigate the effect of evening primrose seed oil (EPO) in stannous chloride *($SnCl_2$)* intoxicated rats. This study was conducted on twenty four albino male rats and classified into four groups, one of them served as control group while the four groups injected with $SnCl_2$. Group 2 received only $SnCl_2$. Groups 3and 4 were administrated 10 and 20 ml/kg/diet/day EPO for 6 weeks. Results revealed that EPO resulted in a significant decrease in aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, tumor necrosis factor, acetyl cholinesteras, very low density lipoprotein, cholesterol and triglycride, creatinine, urea nitrogen and uric acid values, however caused a significant increase in sexual hormonal levels when compared to the control (+ve), in a dose dependant manner. It can be concluded that treatment with of evening primrose oil increases the protective capacity against toxicity effects of stannous chloride.

Introduction

Evening primrose oil *(Oenothera biennis)* is a species of Oenothera native to eastern and central North America. Evening primrose oil *(*EPO) is a good source of the most effective source of gamma-linolenic acid (GLA) among plant and herbs. In addition, the functional activity of consisting of phytochemicals such unsaturated fatty acids **(*Senanayake and Shahidi, 2004 and Peiretti et al., 2004)***. Evening primrose oil is the most important source of seeds known to have anti-atherogenic and hepatoprotective activities, useful for reproductive health, gastrointestinal symptoms immunomodulation and therapeutic advantage over a wide range of disease conditions ***(Hudson, 1984, McFarlin et al., 1999, National Toxicology Program, 2009 and Lisiak et al. 2013).***

Furthermore essential oil of lavender has significant protection against increase of blood glucose as well as enhancement in antioxidant enzymes activities in diabetic rats. Treatment with Evening primrose oil induced a decrease of lipoperoxidation as an increase of antioxidant enzyme activity ***(Blumenthal, 2001).*** EPO is a rich source of the omega-6 essential fatty acids components ***(Dobryniewski et al., 2007 and Natural Standard, 2008).***

Tin is a natural element in the earth's crust. Tin metal is used to line cans for food, beverages, and aerosols. $SnCl_2$ is used in toothpaste, perfumes, soaps, food additives and dyes ***(EC, 2004).*** In addition, stannous chloride, $SnCl_2$, is widely used in daily human life to conserve soft drinks, in food manufacturing and processing packaging ***(EGVM, 2002).*** The biological effects of $SnCl_2$ include stimulation followed by depression of the central nervous system, toxicity of tin chloride, pathological changes in liver, kidney, brain

edema, and pancreatic atrophy ***(Abdollahi et al., 2004 and Aida, et al., 2008).*** The main purpose of the present work was to study the protective effect of EPO on sexual toxicity and biochemical parameters in stannous chloride toxicity in rats.

Materials and Methods

Materials

Evening primrose seed oil: Evening primrose seed oil (EPO) was purchased from the International Herbals Company, Cairo Egypt.

Stannous chloride ($SnCl_2$): Stannous chloride was obtained from Sigma Chemical Co. asere obtained from El-Gomhoriya Pharm. and Chem. Ind. Co. Cairo, Egypt.

Animals: Twenty four male albino rats, *Sprague Dawley* strain, weighing (135 ± 5*g*) were purchased from the animal house of Agriculture Research Center, Giza, Egypt.

Methods:

Antioxidant activity: The effect of evening primrose oil on 1,1-diphenyl-1-picrylhydrazyl DPPH radical was studied, employing the modified method described earlier by ***(Yamaguchi, et al., 1998)*** was calculated using the following equation:

$$\text{Scavenging effect \%} = \frac{\text{Ab. control - Ab Sample (517}nm\text{)}}{\text{Ab control (517}nm\text{)}} \times 100$$

Experimental design:

The experiment was performed in Animal House at the Institute of phthalmology, Giza. All rats were fed for one week prior to the beginning of the experiment on basal diet (BD), then divided into two

main groups, the first group (n= 6 rats) was fed on the BD only as a negative control (–ve) normal rats. The rats of second main group (n= 24 rats) were intraperitoneally injected with $SnCl_2$ at dose of (20 *mg/kg* bwt) 4 times weekly to induce stannous chloride toxicity according to ***(El-Makawy, et al., 2008),*** and fed on basal diet (BD) then divided into 3 sub-groups (each 6 rats) as follows:

Sub-group (2): Injected with $SnCl_2$ were fed on BD without any treatment and considered as a positive control (+ve).

Sub-group (3): Injected rats by $SnCl_2$ were fed on BD containing 10 ml/kg/diet EPO.

Sub-group (4): Injected rats by $SnCl_2$ were fed on BD containing 20 ml/kg/diet EPO**.**

Body weight (BW) was recorded weekly during the experimental period and feed intake was measured daily during the experimental periods. At the end of the experiment, biological evaluation of the tested diets was carried out by determining total feed intake, body weight gain (BWG) and Food efficiency ratio (FER). At the end of the experiment period (6 weeks), blood samples were taken from hepatic portal vein and left to clot by standing.

Biochemical analysis:

Determination of liver enzymes: Serum alanine, aspartate aminotransferases (ALT & AST) and alkaline phosphatase (ALP) was assayed by ***(Reitman and Frankel, 1957 and Kind and King, 1954).***

Determination of kidney functions: Serum creatinine, uric acid and urea were determined according to the methods described by **(Bohmer, 1971**; **Fossati *et al*., 1980 and Patton and Crouch, 1977)**, respectively.

Determination of serum lipids: Enzymatic colorimetric determination of triglycerides and total cholesterol was determined by colorimetric method according to *(**Fossati and Prencipe, 1982 and Allian, et al., 1974)**.* Determination of high density lipoprotein , very low density lipoproteins and low density lipoproteins were carried out according to the method of ***(Fnedewaid, 1972 and Lee and Nieman, 1996).***

Determination of serum acetyl cholinesterase and antioxidant parameters: Acetyl cholinesterase (AchE) activity, superoxide dismutase (SOD) activity, total antioxidants capacity (TAC), and TNF-α were determined according to ***(Knedel and Boottger, 1967, Nishikimi, et al., 1972; Cao, et al., 1993 and Beutler, et al., 1985),*** respectively.

Determination of serum sexual hormonal profile: Serum testosterone, follicle stimulating hormone levels (FSH) and Luteinizing hormone (LH) according to the method described by ***(Wilke and Utley 1987, Ballester et al., 2004 and Schams and Karg (1969).***

Statistical analysis: The obtained data were statistically analyzed using computerized SPSS. Effects of different treatments were analyzed by one way ANOVA (Analysis of variance) test using Duncan's multiple range test and $p<0.05$ was used to indicate significance between different groups ***(Snedecor and Cochran, 1967).***

Results and Disscusion

Radical scavenging activity of EPO

The radical scavenging activity of EPO was measured on 1,1-diphenyl-1-picrylhydrazyl DPPH radical and results were represented in Table 1, data show that EPO recorded (85.9±1.5, 93.7±2.7, 95.6±2.4 and 97.2±1.6 *µg/ml*) at 5, 10, 15 and 20 *min*, respectively.

Body weight gain of stannous chloride intoxicated rats fed on EPO for six weeks Results in Table 2 showed that, untreated stannous chloride intoxicated rats control (-ve) significantly decreased food intake (FI), BWG and FER when compared to control (+ve). Treatment with evening primrose oil (EPO) at levels 10 and 20 *ml/kg* diet, given to stannous chloride intoxicated rats for 6 weeks induced significant increase in BWG and FER compared to control (+ve). These results are in harmony with those obtained by ***(Meehan et al., 1995, Shotton et al., 2004and Borsonelo et al., 2007).***

Effect of EPO on liver function on stannous chloride intoxicated rats

Data recorded in Table 3 reflected the effect of EPO on liver function of stannous chloride intoxicated rats. Positive control group (+ve) recorded significant increase in the activity of liver enzymes AST, ALT and ALP when compared to negative control rats group. Treated groups with EPO decreased liver enzymes in rats compared to control (+ve). It could be observed that liver enzyme levels decreased significantly with the increase of EPO doses in stannous chloride intoxicated rats as recorded (37.07±7.78, 25.07±4.75 and 33.73±4.37 *µ /ml*) for AST, ALT and ALP, respectively in stannous chloride intoxicated rats treated with 10 *ml/kg* diet EPO. Meanwhile, stannous chloride intoxicated rats treated with 20 *ml/kg* diet EPO

recorded (35.02±5.04, 22.68±5.08 and 32.11±3.11 *µ /ml*) for AST, ALT and ALP, respectively.

In the study of ***(Tori Hudson, 2004 and Geppert, et al., 2008)*** they tested the effect of demonstrated that effect of EPO supplementation caused a decrease in haematology and liver enzymes, so adequate EPO intake could play an important role in the prevention and/or modification of different diseases. Data given by ***(Mikešová et al., 2014 and EL-Baz et al., 2015)*** revealed lowest significantly of liver enzymes in hyperlipidemic rats supplemented with 5% EPO, 5% olive oil and 10% Black sesame oil respectively.

Effect of EPO on kidney function on stannous chloride intoxicated rats

Results in Table 4 showed that stannous chloride intoxicated rats had a significant increase in creatinine, urea and uric acid levels (2.67±0.12, 26.93±3.12 and 3.11±0.58 *mg/dl*) compared to the control (-ve) which recorded (1.23±0.16, 15.12±2.09 and 1.68±0.42 *mg/dl*) for the same parameters respectively. Treated groups with evening primrose oil at levels 10 and 20 *ml/kg* diet, given to stannous chloride intoxicated rats for 6 weeks significantly decreased the elevated serum levels of creatinine, urea nitrogen and uric acid levels when compared to the control (+ve), in a dose dependant manner.

It could be observed that 20 *ml/kg* diet of EPO enhanced kidney functions of stannous chloride intoxicated rats as recorded the lowest values of creatinine 1.37±0.21 *mg/dl*, urea 18.67±3.06 *mg/dl* and uric acid 1.82±0.18 *mg/dl* values comparing with all stannous chloride intoxicated rats groups

In a study performed by ***El-Baz, et al., (2015),*** hyperlipidemic rats showed a significant increase in uric acid ancreatinine levels with percentage increase of 32.75 and 50.55%, respectively. However, co administration of hyperlipidemic diet with 5% EPO showed an improvement in percentages of uric acid and creatinine levels 24.01%, for uric acid and 73.62%, for creatinine. These data are confirmed with ***(Barcelli et al., 1986, Burgess et al., 1995 and Papanikolaou et al., 1996).*** These data may be due to the biochemical properties of EPO which acts as antioxidant agent and its pharmedynamics and pharmakinetics actions on the healing of residual renal function.

Effect of EPO on serum lipid profile on stannous chloride intoxicated rats

As demonstrated in Table 5 stannous chloride intoxicated rats had a significant increase in the serum levels of total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL-c), very low density lipoprotein (VLDL-c) while, there was a significant decrease in serum level of high density lipoprotein (HDL-c) when compared to those fed on BD only (-ve control).

However treatment with evening primrose oil at levels 10 and 20 *ml/kg* diet, given to stannous chloride intoxicated rats for period significantly decreased the elevated serum levels of TC (90.67±11.53, 83.33±12.08 *mg/dl*), TG (119.07±9.40, 110.00±11.20 *mg/dl*), LDL-c (35.06±8.02, 32.07±9.06 *mg/dl*) and VLDL-c (23.53±1.81, 22.00±1.31 *mg/dl*), increased serum levels of HDL-c (28.13±4.11, 29.89±3.53 *mg/dl*) for 10 and 20 *ml/kg* diet of EPO stannous chloride intoxicated rats, respectively when compared to control (+ve), in a dose dependant manner. Treatment with EPO in this study has important role to increase of zinc concentration. Lipid

lowering effect in present study might be due to anti-atherogenic effect of vitamin E, gamma-linolenic acid (GLA) and antioxidizing effect of high phenolic contents of EPO as reported by ***(Fukushima et al., 1997, Cintra et al., 2006 Peschel et al., 2007, Ghasemnezhad and Honermeier, 2007 and Riaz et al., 2009).***

EPO can prevent changes in plasma lipid, which linked with being rich in linoleic acid, an essential unsaturated fatty acid. The protective effect is related to abundant linoleic acid and oleic acid, these oils were good for reducing serum cholesterol, triacylglycerides and LDL and increase HDL levels which associated with reducing risk of heart attack ***(Villalobos et al., 1998; Horrobin and Manku, 1983; Sugano et al., 1986; Kim et al. 2006; Lee et al., 2006 and Magda and Afifi 2011)***

Effect of EPO on antioxidant parameters on stannous chloride intoxicated rats

As shown in Table 6, a significant decreased in the levels of total antioxidants capacity (TAC), TNF-α and superoxide desmutase (SOD) (1.17±0.15 *mmol/L*, 3.02±0.09 *pg/ml* and 0.26±0.09 *mg/L*) respectively, while a significant increase in the levels of acetyl cholinesterase (AchE) 164.70±25.75 *U/L* was observed in control group (+ve) when compared to control (-ve) and stannous chloride intoxicated rats groups treated with 10 and 20 *ml/kg* diet of evening primrose oil.

It noticed that the administration of EPO at levels 10 and 20 *m/kg* diet, given to stannous chloride intoxicated rats for 6 weeks significantly decreased AchE and significantly increased total antioxidants levels and enhanced activity of SOD compared to

positive control rats group, in a dose dependant manner. The crude EPO extract contains high levels of antioxidants, including tocopherols ***(Eskin, 2008)***. They explained that anti-inflammatory compounds of flavonoids that present in EPO inhibited expression ***(Benatti et al., 2004).*** It is also an antioxidant that helps to support the body's functions and maintain them in a normal range by neutralizing free radicals ***(Christie, 1999).***

The results in this study was similar to that observed by **Evans** ***& Goldfine, 2000; Gofron et al., 2006; Bekyarova et al., 2007; El-kossi et al. (2011)*** they attributed the beneficial activity to the high content of phenloic phytochemicals which present in the EPO. ***De La Cruz et al., (1999) and Mikešová et al., (2014)*** indicate that EPO as a very important antioxidant effectively reduced the rate of oxidative stress generated during the workload of horses racehorses.

Effect of EPO on sexual hormonal parameters on stannous chloride intoxicated rats

Effects of evening primrose oil on sexual hormonal levels on stannous chloride intoxicated rats groups are presented in Table 8. A significant decreased in the level of testosterone, follicle stimulating and luteinizing hormones (10.03±3.01, 91.17±7.55 and 1.35±1.16 *ng/mL*) respectively was observed in control (+ve) when compared to control (-ve) and stannous chloride intoxicated rats groups treated with 10 and 20 *ml/kg* diet of EPO. Treatment with EPO at levels 10 and 20 *ml/kg* diet, given to stannous chloride intoxicated rats for 6 weeks significantly increased sexual hormonal levels when compared to the control (+ve) as recorded (18.12±3.01, 115.98±9.65 and 2.92±0.96 *ng/mL*) for testosterone, follicle stimulating and luteinizing hormones, respectively in rats group fed on 10 ml/kg diet of EPO,

while the same hormones recorded (21.81±9.64, 119.59±9.80 and 3.09±0.96 *ng/mL*) respectively in rats group fed on 20 *ml/kg* diet of EPO.

EPO is an omega-fatty-acid-6 rich oil, containing both LA and GLA which produce prostaglandin. Prostaglandins are used to regulate hormone production and funcitioning ***Tori Hudson, (2004), Leaver et al. (1986) and Shin and Lee (2006),*** they reported that EPO can used as antioxidant, which have rich source of fatty acids and phytochemical compounds, which promote prostate health by increasing serum testosterone, thus improving reproductive functions.

Conclusion

It could be concluded that, amelioration of lipid profile, hepato-renal function, antioxidant capacity as well as enhancing sexual hormones were detected in response to EPO against toxicity effects of stannous chloride.

Table (1): Effects of evening primrose oil on DPPH radical

Evening primrose seed oil	5 (µg/ml)	10(µg/ml)	15(µg/ml)	20(µg/ml)
Scave	85.9±1.5	93.7±2.7	95.6±2.4	97.2±1.6*

Table (2): Effect of evening primrose oil on food intake (FI), body weight gain (BWG) and food efficiency ratio (FER) on stannous chloride intoxicated rats.

Parameters / Groups	BWG (*g*)	FI (*g/day*)	FER
-ve control group	139.3±8.1 [a]	16.5±0.58 [a]	0.188±0.04 [a]
+ve control group	98.8± 5.3 [c]	12.6±0.58 [c]	0.137±0.01 [c]
10 ml EPO	128.4±7.3 [b]	16.3±0.37 [b]	0.182±0.00 [b]
20 ml EPO	136.0±7.8 [b]	16.4±0.45 [b]	0.187±0.01 [b]

EPO: Evening primrose oil; Mean ± SD with the same letter is insignificantly different.

Table (3): Effect of evening primrose oil on serum levels of liver enzymes; aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) on stannous chloride intoxicated rats

Parameters / Groups	AST (µ /ml)	ALT (µ /ml)	ALP (µ /ml)
-ve control group	29.33±5.53[c]	18.63±5.15[c]	29.17±5.66 [c]
+ve control group	41.33±4.05[a]	30.08±4.29[a]	47.38±5.81 [a]
10 ml EPO	37.07±7.78[b]	25.07±4.75[b]	33.73±4.37 [b]
20 ml EPO	35.02±5.04[c]	22.68±5.08[b]	32.11±3.11 [c]

EPO: Evening primrose oil; Mean ± SD with the same letter is insignificantly different.

Table (4): Effect of evening primrose oil on serum levels of creatinine, urea and uric acid on stannous chloride intoxicated rats

Parameters / Groups	Creatinine (mg/dl)	Urea (mg/dl)	Uric acid (mg/dl)
-ve control group	1.23±0.16 ^{c}	15.12±2.09 ^{c}	1.68±0.42 ^{b}
+ve control group	2.67±0.12 ^{a}	26.93±3.12 ^{a}	3.11±0.58 ^{a}
10 ml EPO	1.68±0.11 ^{b}	20.33±2.08^{b}	2.56±0.03 ^{b}
20 ml EPO	1.37±0.21 ^{b}	18.67±3.06 ^{bc}	1.82±0.18 ^{b}

EPO: Evening primrose oil; Mean ± SD with the same letter is insignificantly different.

Table (5): Effect of evening primrose oil on serum total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL-c), low density lipoprotein (LDL-c) and very low density lipoprotein (VLDL-c) on stannous chloride intoxicated rats

Parameters / Groups	TC (mg/dl)	TG (mg/dl)	HDL-c (mg/dl)	LDL-c (mg/dl)	VLDL-c (mg/dl)
-ve control group	77.00± 9.07^{d}	97.00± 9.80^{d}	38.07± 1.53^{a}	24.60± 5.17^{b}	20.12± 1.20^{c}
+ve control group	110.67± 9.79^{a}	176.01± 13.67^{a}	23.03± 4.21^{d}	41.08± 7.89^{a}	36.01± 1.72^{a}
10 ml EPO	90.67± 11.53^{b}	119.07± 9.40^{b}	28.13± 4.11^{c}	35.06± 8.02^{c}	23.53± 1.81^{b}
20 ml EPO	83.33± 12.08^{c}	110.00± 11.20^{c}	29.89± 3.53^{b}	32.07± 9.06^{c}	22.00± 1.31^{b}

EPO: Evening primrose oil; Mean ± SD with the same letter is insignificantly different.

Table (6): Effect of evening primrose oil on antioxidant parameters and acetyl cholinesterase on stannous chloride intoxicated rats

Parameters / Groups	Antioxidants capacity (mmol/L)	Tumor necrosis factor (pg/ml)	Superoxide dismutase (mg/L)	Acetyl cholinesterase (U/L)
-ve control group	2.45± 0.22 [a]	5.77± 1.33 [c]	1.73± 0.12[a]	140.73± 13.61 [d]
+ve control group	1.17± 0.15 [c]	3.02± 0.09 [a]	0.26± 0.09[d]	164.70± 25.75 [a]
10 *ml* EPO	1.98± 0.15 [b]	4.57± 1.26 [b]	1.08± 0.02 [c]	140.00± 14.58 [b]
20 *ml* EPO	2.24± 0.06 [b]	5.40± 1.09 [bc]	1.62± 0.07 [b]	144.83± 14.75[c]

EPO: Evening primrose oil; Mean ± SD with the same letter is insignificantly different.

Table (7): Effect of evening primrose oil on sexual hormonal parameters on stannous chloride intoxicated rats

Parameters / Groups	Testosterone hormone (ng/mL)	Follicle Stimulating Hormone (ng/mL)	Luteinizing hormone (ng/mL)
-ve control group	22.2±6.7 a	122.38±10.75 a	3.68±0.76 a
+ve control group	10.03±3.01 c	91.17±7.55 c	1.35±1.16 c
10 ml EPO	18.12±3.01 b	115.98±9.65 b	2.92±0.96 b
20 ml EPO	21.81± 9.64 b	119.59±9.80 b	3.09±0.96 b

EPO: Evening primrose oil; Mean ± SD with the same letter is insignificantly different.

References

Abdollahi, M.; Ranjbar, A.; Shadnia, S.; Nikfar, S. and Rezaie, A. (2004):
Pesticides and oxidative stress: A review. Med. Sci. Monit.; 10: RA141-RA147.

Aida I. El-Makawy, Shenouda M. Girgis, and Wagdy K.B. Khalil (2008):
Developmental and genetic toxicity of stannous chloride in mouse dams and fetuses. Mutation Research 657: 105–110.

Allian, C; Poon, L; Chan, C and Richmond, W (1974):
Enzymatic determination of total serum cholesterol. Clin. Chem., 20: 470.

Ballester, J; Munoz, MC; Dominguez, J; Rigau, T; Guinovart, JJ and Rodriguez, JE (2004):
Insulin-dependent diabetes affects testicular function by FSH – linked mechanisms. *J.Androl*., 25(5): 706-719.

Barcelli, UO; Miyata, J and Ito, Y (1986):
Beneficial effects of polyunsaturated fatty acids in partially nephrectomized rats. Prostaglandins, 32:211-9.

Bekyarova, GY; Ivanova, DG and Madjova, VH (2007):
Molecular mechanisms associating oxidative stress with endothelial dysfunction in the development of various vascular complications in diabetes mellitus. *Folia Med (Plovdiv)*., 49(3-4):13-9.

Benatti, P; Peluso, G; Nicolai, R and Calvani, M (2004):
Polyunsaturated fatty acids: biochemical, nutritional and epigenetic properties. Journal of the American College of Nutrition, 23, 281–302.

Beutler, B; Greenwald, D and Hulmes, JD (1985):
Identity of tumour necrosis factor and the macrophage-secreted factor cachectin. *Nature*, 316:552-554.

Bohmer, H (1971):
Micro-determination of creatinine. *Clin. Chem. Acta.,* 32:81-85.

Borsonelo, EC; Galduróz, JCF; Suchecki, D and Calil, HM (2007):
The influence of n-6 fatty acid supplemented diet on the effect of imipramine in an animal model of depression. Pharmacol Biochem Behav, 86(1):113-116.

Burgess, NA; Reynolds, TM; Williams, N; Pathy, A and Smith, S (1995):
Evaluation of four animal models of intrarenal calcium deposition and assessment of the influence of dietary supplementation with essential fatty acids on calcification. Urol Res., 23:239-42.

Cao, G; Alessio, H and Cutler, R (1993):
Oxygen radical absorbance capacity assay for antioxidants. Free Radic Biol Med., 14:303-311.

Christie, WW (1999):
The analysis of evening primrose oil. Industrial Crops and Products, 10, 73–83.

Cintra DEC; Costa AGV; Peluzio MCG; Matta, SLP; Silva MTC and Costa, NMB (2006):
Lipid profile of rats fed high-fat diets based on flaxseed, peanut, trout, or chicken skin. *Nutrition,* 22:197-205.

De La Cruz, JP; Quintero, L; Galvez, J; Villalobos, MA and Sanchez de la Cuesta F (1999):
Antioxidant potential of evening primrose oil administration in hyperlipemic rabbits . *Life Sci*., 65:543-55.

Dobryniewski, J; Szajda, SD; Waszkiewicz, N and Zwierz K (2007):
The gamma-linolenic acid (GLA)--the therapeutic value. Przegl Lek 2007;64(2):100-2.

EC (European Commission) (2004):
Commission Regulation (EC) No 242/2004 of 12 February 2004 amending Regulation (EC) No 466/2001 as regards inorganic tin in foods. Officfial Journal of the European Union L 42/3, 13.2.2004.

EGVM (Expert Group on Vitamins and Minerals) (2002):
Revised review of tin. Technical report on the carcinogenesis bioassay if stannous chloride (CAS No 7772-99-8) in F3444.N rats and B6C3F1N mice (feed study). NIH, PUB-82-1787. 1-158, NTP, Research Triangle Park, NC, USA.

EL-Baz, FK; SALAMA, Z; ALY, HF and HANAN A and TAIE, HA (2015):
Potency of sesame oil as antihypercholesterolemic agent in rats fed high-fat diet. *Int J Pharm Bio Sci.,* 6(3): (B) 177 – 189.

El-kossi, AA; Abdellah, MM; Rashad; AM and Hamed, SA (2011):
The effectiveness of evening primrose oil and alpha lipoic acid in recovery of nerve function in diabetic rats; 2 (3): 245-253.

El-Makawy, Al; **Girgis, SM and Khalil, WK (2008):**
Developmental and genetic toxicity of stannous chloride in mouse dams and fetuses. Mutat Res. Mrgentox.

Eskin, NAM (2008):
Borage and evening primrose oil. *Eur J Lipid Sci Technol*, 110(7):651-654.

Evans, JL and Goldfine ID (2000):
α-Lipoic acid: a multifunctional antioxidant that improves insulin sensitivity in patients with type 2 diabetes. Diab Techn Therap., 2(3):401-13.

Fnedewaid, WT (1972):
Determination of HDL. Clin. Chem., 8:499.

Fossati, P; Prencipe, L and Berti, G (1980):
Use of 3.5 dichloro–z–hydroxybenzenesulfonic acid / 4 aminophenazone chromogenic systems in direct enzymic assay of uric acid in serum and urine. Clin. Chem.; 26:227-231.

Fossati, P and Prencipe, L (1982):
Determination of triglycerides, Bicon Diagnostics, made in Germany. Clinical Chemistry., 28: 2077-2078.

Fukushima, M; Matsuda, T; Yamagishi, K and Nakano, M (1997):
Comparative hypocholesterolemic effects of six dietary oils in cholesterol-fed rats after long-term feeding. Lipids 1997; 32:1069-74

Geppert, J; Demmelmair, H; Hornstra, G and Koletzko, B (2008):
Co-supplementation of healthy women with fish oil and evening primrose oil increases plasma docosahexaenoic acid, g -linolenic acid and dihomo- g - linolenic acid levels without reducing arachidonic acid concentrations. British Journal of Nutrition, 99:360–369

Ghasemnezhad, A and Honermeier, B (2007):
Seed yield, oil content and fatty acid composition of oenothera biennis L. affected by harvest date and harvest method. Industrial Crops and Products, 25: 274-281.

Gofron, MS; Bellwon, J; Sobiczewski, W; Ciecwierz, D; Rynkiewicz, A and Stolyhwo, A (2006):
Two weeks supplementation with olive oil and evening primrose oil similarly affect plasma lipids level in men with coronary artery disease. Nutritional Approaches; XIV international Symposium on Atherosclerosis, 18-22.

Horrobin, DF and Manku, MS (1983):
How do polyunsaturated fatty acids lower plasma cholesterol levels? Lipids 18:558-562.

Hudson, B J F (1984):
Evening primrose (Oenothera spp.) oil and seed. JAOCS 61:540-543. Blumenthal M. Herb sales down 15% in mainstream market. HerbalGram 2001; 51:69.

Kim, JJ; Yu, SH; Jeon, WM and Kwak, HS (2006):
The Effect of Evening Primrose Oil on Chemical and Blood Cholesterol Lowering Properties of Cheddar Cheese. Asian-Aust. J. Anim. Sci., 19 (3): 450-458.

Kind, PR and King, EJ (1954):
Estimation of alkaline phosphatase activity by determination of hydrolyzed phenol with aminoantipyrene. J. Clin.Path.7: 322.

Knedel, M and Boottger, R (1967):
A kinetic method for determination of the activity of pseudo cholinesterase. Klin. Wochenschr., 45: 325-327.

Leaver, HA; Lytton, FD; Dyson, H; Watson, ML and Mellor, DJ (1986):
The effect of dietary ω 3 and ω 6 polyunsaturated fatty acids on gestation, parturition and prostaglandin E 2 in intrauterine tissues and the kidney. Prog Lipid Res, 25:143-146.

Lee, R and Nieman, D (1996):
Nutritional assessment. 2 nd Ed., Mosby, Missoun, USA.

Lee, SJ; Hwang, JS; Lee, S; Ahn, J and Kwak, HS (2006):
Property changes and cholesterol lowering effect in evening primrose oil-added and cholesterol-reduced yogurt. Int. J. Dairy Technol., (in press).

Leung A and Foster S (1996):
Encyclopedia of Common Natural Ingredients used in Food, Drugs and Cosmetics, 2nd edition, John Wiley & Sons, Inc., 235-8.

Lisiak, D; Grzeskowiak, E; Borzuta, K; Raj S; Janiszewski, P and Skiba, G (2013):
Effects of supplementary vegetable and animal fats on the slaughter values of fatteners, meat quality, and fatty acid profile in pigs. Czech Journal of Animal Science, 11, 497–511.

Magda, SM and Afifi , AA (2011):
Effect of some polyunsaturated fatty acids rich-oils on plasma lipid profiles using normal rats. J.Agric. Chem. and Biotechn., Mansoura Univ., 2 (2): 49 – 59.

McFarlin, BLM; Gibson, MH; O'Rear, J and Harman, P (1999):
A National survey of herbal prepration use by Nursemidwives for labor stimulation. Journal of Nurse-Midwifery, 44: 205-216.

Meehan, ED; Beaugé, F; Choquart, D and Leonard, BE (1995):
Influence of an n-6 polyunsaturated fatty acid-enriched diet on the development of tolerance during chronic ethanol administration in rats. Alcohol Clin Exp Res, 19(6):1441-1446

Mikešová1, K; Härtlová, H; Zita, L; Chmelíková, E; Hůlková, M and Rajmon, R (2014):
Effect of evening primrose oil on biochemical parameters of thoroughbred horses under maximal training conditions. Czech J Anim. Sci., 59, (10): 488–493.

National Toxicology Program (2009):
Chemical Information Review Document for Evening Primrose Oil (Oenothera biennis L.) [CAS No. 90028-66-3] Supporting Nomination for Toxicological Evaluation.

Natural Standard (2008):
Evening primrose oil (Oenothera biennis L.); monograph. Internet address: http://www.naturalstandard.com.

Nishikimi, M; Rao, N and Yogi, K (1972):
Colorimetric determination of superoxide dismutase. Biochem. Biophys. Res. Common., 46: 849-854.

Patton, C. and Crouch, S. (1977):
Determination of serum urea enzymatically. J. of Ana. Chem.; 49 : 464 - 469.

Papanikolaou N, Darlametsos I, Tsipas G (1996):
Effects of OKY-046 and nifedipine in cyclosporineinduced renal dysfunction in rats. Prostaglandins Leukot Essent Fatty Acids , 55:249-56.

Peiretti, BAPG; Palmegiano, GB and Masoero, G (2004): Chemical composition, organic matter digestibility and fatty acid content of evening primrose (Oenothera paradoxa) during its growth cycle. Animal Feed Science and Technology, 116: 293-299.

Peschel, W; Dieckmann, W; Sonnenschein, M and Plescher, A (2007):
High antioxidant potential of pressing residues from evening primrose oil in comparison to other oilseed cakes and plant antioxidants. Industrial crops and products, 25: 44-50.

Reitman, S and Frankel, S (1957):
Determination of serum alanine and aspartate aminotransferases (ALT & AST)**.** Clin .Path. Am. J., 28: 57-63.

Riaz, A; Khan, RA and Ahmed, SP (2009):
Assessment of anticoagulant effect of evening primrose oil. Pak J.Pharm.Sci., 22: 355-359.

Schams, D and Karg, H (1969):
Studien über die Spezifität des Radioimmunotests zur Bestimmung des Luteinisierungshormons im Rinderblut. Hoppe-Seyhr's Z- physiol. Chem., 351, 41.

Senanayake, SPJN and Shahidi, F (2004):
Incorporation of docosahexaenoic acid (DHA) into evening primrose oil (Oenothera biennis L.) oil via lipase-catalyzed transesterification. Food Chemistry, 85: 489- 496.

Shin, SJ and Lee, JH (2006):
Improving effect of evening primrose oil on the sexual functions of male mice. Korean J Pharmacogn, 37(2):85-91.

Shotton, HR; Broadbent, S and Lincoln, J (2004):
Prevention and partial reversal of diabetes-induced changes in enteric nerves of the rat ileum by combined treatment with α-lipoic acid and evening primrose oil. Autonomic Neuroscience: Basic and Clinical, 111: 57-65.

Snedecor, GW and Cochran, WG (1967):
Statistical Methods; 7th Ed., The Lowa State University Press., Ames, Lowa, U.S.A.

Sugano, M; Ide, T; Ishida, T and Yoshida, K (1986):
Hypocholesterolemic effect of gamma-linolenic acid as evening primrose oil in rats. Ann. Nutr. Metabol., 30:289-299.

Tori Hudson, N D (2004):
Evening Primrose Oil in Natural Medicine 7. 3(5).

Villalobos, MA; De, La; Cruz, JP; Martin-Romero M; Carmona JA; Smith-Agreda JM; Sanchez de la Cuesta F (1998):
Effect of dietary supplementation with evening primrose oil on vascular thrombogenesis in hyperlipemic rabbits. Thromb Haemost, 80:696-701.

Wilke, TJ and Utley, DJ (1987):
Total testosterone, free androgenic index and calculated free testosterone by analog RIA method. Clinc. Chem.;33:1372:1375.

Yamaguchi, T; Takamura, H; Matoba, T and Terao, J (1998):
HPLC method for evaluation of the free radical-scavenging activity of foods by using 1, 1,-diphenyl-2-picrylhydrazyl. Bioscience Biotechnology Biochemistry, 62: 1201-04.

تأثير زيت بذور زهرة الربيع المسائية ضد السمية الناتجة عن كلوريد القصدير في ذكور الفئران

هناء فاروق المهيرى – منى ياسر عبد الخالق مصطفى

قسم الاقتصاد المنزلى- كلية التربية النوعية – جامعة المنصورة- مصر

الملخص العربي

يستخرج زَّيت زهرة الربيع المسائيّة من بذور النبات لما له من فوائد وأهمية كمضادة للأكسدة والالتهابات. وأجريت هذه الدراسه لمعرفة الآثار المترتبه على تناول زيت بذور زهرة الربيع المسائيه ضد السمية الناتجة عن كلوريد القصدير في ذكور الفئران . وقد أجريت هذه الدراسة على أربعة وعشرين ذكور الفئران البيضاء وقسمت إلى اربع مجموعات المجموعة الاولى الضابطه السالبة، بينما المجموعات الثالثة الأخرى فتم حقنها بمركب كلوريد القصدير وتركت مجموعة واحدة كمجموعة (الضابطة موجبة) غير معالجة. بينما المجموعات الأخرى عولجت بمستويات مختلفة من زيت زهرة الربيع 10 و 20 مللي / كجم من الوجبة/ يوميا) لمدة ستة أسابيع.

وقد أظهرت النتائج تأثير زيت زهرة الربيع على الفئران كمضاد للأكسده مع التنشيط لمستوى الهرمونات الجنسية الذكورية وكذلكً وظائف الكبد والكلى ومستوى دهون الكلى، وقد أدى تناول الفئران للزيت بجرعات مختلفة لمدة ستة أسابيع إلى خفض كلا من أنزيمات الكبد وعامل نخر الورم والاسيتيل كولين استريز والكولستيرول والدهون الثلاثيه وحمض اليوريك والكرياتينين في التسمم الناتج بكلوريد القصدير فى الفئران مقارنة بالمجموعة الضابطة الموجبة للفئران. ووتوصى الدراسة القدره الوقائية لزيت زهرة الربيع المسائيه ضد التأثير السام لكلوريد القصدير.